34 | Chest Roentgenography for Cardiovascular Evaluation

ERIC MANHEIMER

Definition and Basic Science

These topics are presented in Chapter 48, Chest Roentgenography for Pulmonary Evaluation.

Technique and Clinical Significance

Despite the ever-increasing number of new diagnostic imaging techniques available to today's clinician, the chest x-ray remains a simple, easy, inexpensive, and most informative examination. In some areas it has been replaced by more sensitive techniques; for example, sonography in the evaluation of pericardial effusions. Still, the chest roentgenogram provides invaluable information as part of the crucial first step in differential diagnosis and in following progression of disease. An appreciation of normal, abnormal, and normal variants in cardiovascular anatomy is essential. A systematic approach is presented that sequentially examines (1) heart size and shape, (2) pulmonary vasculature (lung fields and hilum), (3) aorta, and (4) thoracic cage. Figure 34.1 shows a normal posteroanterior (PA) view of the chest.

Figure 34.1
A normal PA chest x-ray. The silhouette borders are (1) right atrium, (2) location of azygous vein (not present on erect film except with increased right-sided cardiac pressures), (3) superior vena cava, (4) aortic knob, (5) left hilum, (6) left atrial appendage, (7) left ventricle.

The Cardiac Silhouette

An enlarged heart is indicative of cardiac disease, but a "normal"-size heart does not imply normality; for example, significant hypertrophy does not cause dilatation and relative enlargement may not be appreciated without comparison films. A "normal" 12 cm heart is not normal if it measured 10 cm 6 months ago. Among many available measurements, the *cardiothoracic ratio* (or CT ratio) benefits from simplicity and utility. It is measured by dividing the maximal transverse cardiac silhouette by the maximal internal thoracic diameter using 0.5 as the upper limit of normal. This will give some false positives, particularly if a film is taken on a poor inspiration. The degree of inspiration has the greatest effect on apparent heart size. An optimal film would have the diaphragm at the posterior tenth rib at maximal inspiration to eliminate "pseudocardiomegaly" and pulmonary plethora. Other causes of a false impression of cardiomegaly are the greater magnification of the heart on an AP film. A good estimate of cardiomegaly is a heart greater than 15 cm in transverse diameter or greater than 1 to 2 cm increase in size over one year.

The identification of individual chamber enlargement is fraught with many difficulties, and it is often impossible to specify which chamber is enlarged. Certain things can be ascertained with relative ease. On a PA view, the right heart border is formed by the right atrium, and the left heart border is formed by the appendage of the left atrium superiorly and the left ventricle inferiorly. On the lateral view, the anterior cardiac border is formed by the right ventricle and the posterior heart border by the left atrium superiorly (just below the carina) and left ventricle inferiorly. Enlargement of individual chambers is usually reflected in a globular bulge in the expected position of that chamber; for example, the right ventricle will expand and fill the retrosternal space on the lateral view. With massive *right ventricular enlargement* there will be additional rotation of the heart. This causes a portion of the right ventricle to show as the right heart border and the left ventricular silhouette to disappear (i.e., it is rotated posteriorly) on the PA view. In the adult, the cause of isolated right ventricular enlargement is usually cor pulmonale, and therefore the pulmonary outflow tract and often the central hilar vessels are also enlarged.

Left ventricular enlargement expands the left heart border on the PA view (Figure 34.2) and the inferior posterior border on the lateral. This is probably the most common isolated chamber enlargement in adults. It occurs frequently with ischemic heart disease and systemic hypertension. It tends to give the heart a "boot-shaped" appearance on the PA view. However, there can be significant hypertrophy or restrictive cardiomyopathy without any chamber enlargement.

Left atrial enlargement is reflected earliest by enlargement of the appendage on the PA view. More difficult to see is the posterior bulge on the lateral view just below the carina.
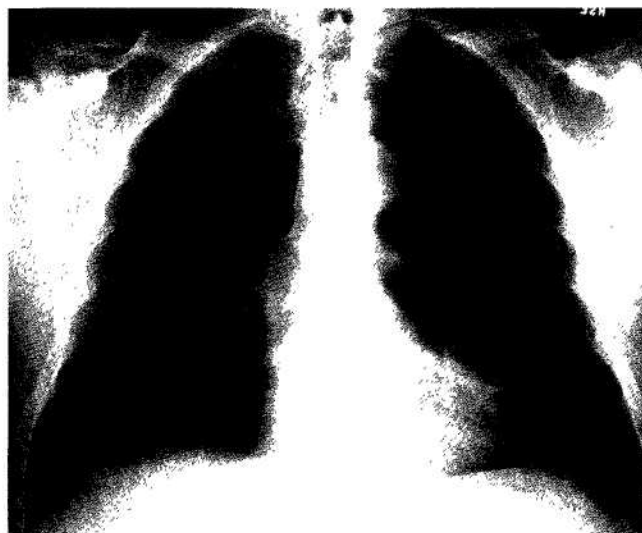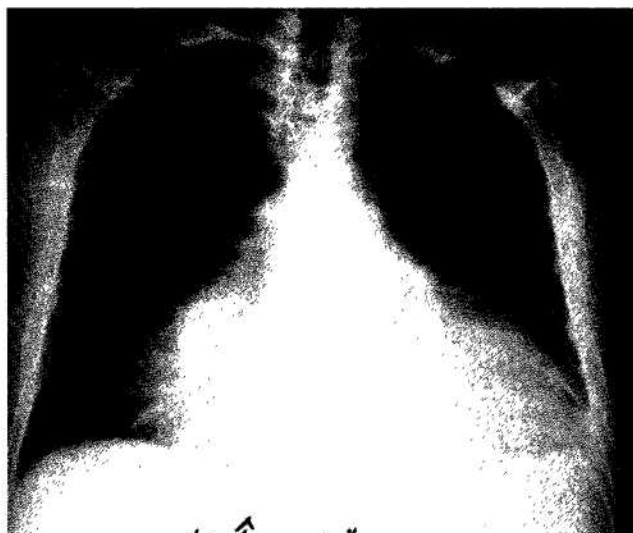
Figure 34.2
Left ventricular enlargement tends to round the left heart border as it enlarges laterally and downward.

Figure 34.3
The classic "water bottle" heart is nonspecific for pericardial effusion and can be seen in cardiomyopathy. Sonography is the most sensitive diagnostic technique in diagnosing an effusion.

This is often obscured by slight patient rotation and the hilar vessels. Other classic but not as often seen signs of left atrial enlargement are the double density and widening of the angle of the carina on the PA view. The double density is the shadow of the left atrium seen through the right atrial shadow just below and to the right of the carina on the PA view. The normal carinal angle is said to be 50 to 100 degrees, a more obtuse angle reflecting left atrial enlargement. However, a poor inspiratory excursion is also a cause of an increased angle, making this sign of limited usefulness. A prominent left atrial appendage on the PA view is probably the most consistent sign of enlargement. A common cause of isolated left atrial enlargement is mitral valve disease. Often, if there is significant left atrial enlargement, there will be associated changes in the lung fields. Left ventricular enlargement (generally from ischemic or hypertensive heart disease), when it progresses to early cardiac decompensation, is often accompanied by left atrial enlargement.

Generalized cardiac enlargement with a globular or water-bottle shape (Figure 34.3) indicates either pericardial effusion or cardiomyopathy. The two cannot be differentiated on chest x-ray except by the infrequently seen pericardial fat sign on the lateral view. Occasionally, the pericardial fat is seen as a thin, lucent crescent between the soft tissue density of the myocardium and the effusion. It is seen on the lateral view behind the sternum in the region of the right ventricle. Echocardiography is a more reliable way to detect a pericardial effusion. Even small effusions can interfere with right heart filling and the chest x-ray will not be abnormal until at least 200 ml of fluid have accumulated.

Constrictive pericarditis is difficult to diagnose on chest x-ray as it is not routinely associated with either a large or small heart. Additionally, pericardial calcification is not diagnostic of constriction. It can be seen as the sequelae of infection or surgery (e.g., pericardial window).

In general, *cardiac calcifications,* whether valvular, pericardial, myocardial, or coronary arterial, are best seen by fluoroscopy. The intrinsic motion of the heart improves visualization on fluoroscopy and obscures visualization on plain films. When pericardial calcification is seen on plain films, it is best visualized on the lateral view as a circular or crescentic calcification surrounding the heart. It may involve the entire heart shadow or only a small portion. Valvular calcium is also best seen on the lateral view. If the heart is bisected along its long axis on the lateral view, aortic valve calcification will lie anterior to this line and mitral calcification posterior (Figure 34.4). Coronary artery calcification is seen anywhere along the expected course of the arteries. The left anterior descending and circumflex arteries are the easiest to see and, again, are best seen on the lateral view. Coronary artery calcification is infinitely easier to see on fluoroscopy and indicates significant coronary artery disease.

Pulmonary Vasculature

Evaluation of the pulmonary vasculature provides vital information about cardiac function. Interpretation of these changes is sometimes made difficult by other noncardiac diseases that also have an effect on the lung parenchyma and pulmonary vessels. For example, emphysema, by destroying normal pulmonary architecture, can alter the typical symmetric infiltrates of pulmonary edema. Basically, three pathophysiologic states can be differentiated: (1) pulmonary venous hypertension or postcapillary hypertension, most notable in congestive heart failure where there is a passive increase in pulmonary pressure through a valveless system; (2) pulmonary arterial hypertension or precapillary hypertension, which is the end product of many disease states with markedly different pathophysiologies; and (3) shunting due to congenital heart diseases.

Interpreting the presence of pulmonary venous hypertension or postcapillary hypertension involves assessing the peripheral vessels within the lung fields and the central or hilar vessels, including not only the pulmonary arteries and veins but also the azygous vein and superior vena cava. Normal peripheral vessels exhibit an orderly branching pattern and a progressive decrease in size. On the erect film, the lower lobe vessels are larger (i.e., wider in diameter)

Figure 34.4
The standard lateral film is bordered by (1) the right ventricle and (2) the left ventricle. To determine the origin of visible calcification within the cardiac silhouette, an imaginary line bisecting the long axis places aortic calcifications above and mitral below.

Figure 34.5
This radiograph represents florid interstitial pulmonary edema with numerous short parallel horizontal lines abutting the pleura in stepladder fashion, or Kerley B lines.

than the upper lobe vessels. The lower lobe vessels are larger at the bases since they contain a greater blood flow from a hydrostatic pressure of approximately 30 cm H_2O compared with 0 cm H_2O at the apices. With early left-sided failure, there is greater flow to the upper lobes. This is called *cephalization of blood flow*. Normally the upper lobe vessels are smaller in caliber than their lower lobe counterparts. With cephalization, the upper lobe vessels become the same size or are larger than the lower lobe vessels. There are other causes of cephalization such as emphysema and a supine patient. This latter gravity-dependent phenomenon makes interpretation of cephalization on a portable film difficult. The key is to compare portable films with each other and not with an erect PA film.

Another early sign of pulmonary venous hypertension is *engorgement of the central veins*, that is, the azygous vein and the superior vena cava. The azygous vein is located in the proximal convex curve of the right upper lobe bronchus. The superior vena cava is a faint vertical shadow to the right of the trachea. These are not always visible in the normal patient but will reliably dilate and become visible in early cardiac failure.

These early changes of cephalization and central venous enlargement may occur before auscultatory changes are present. This is particularly useful in acute myocardial infarction where pulmonary edema may not yet be clinically evident.
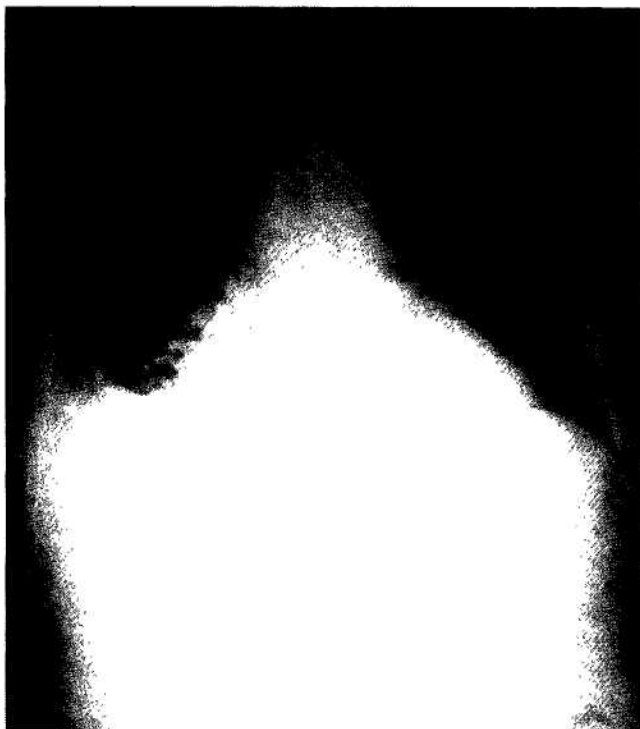
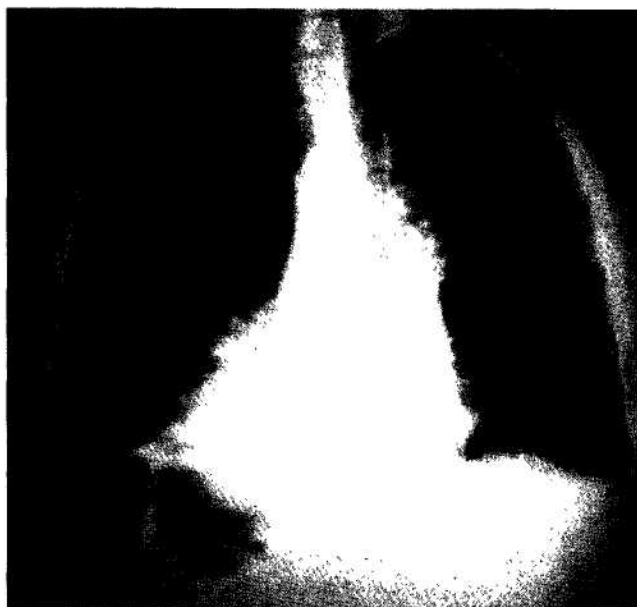As the pressure increases in pulmonary venous hypertension, the engorged veins leak fluid across their capillary membranes, first into the interstitium of the lung, and, finally, with increasing pressure, into the alveoli. The first phase, interstitial edema, has many varied appearances. As the fluid fills the interstitium, it causes a nonspecific loss of the usually sharp vascular markings (particularly about the hilum, called *perihilar haziness)*, increased thickness of the bronchial walls seen end-on, usually near the hilum (*peribronchial cuffing*), and the appearance of short, thin, 1 to 2 cm long parallel lines seen at right angles to the pleura, laterally at the lung bases, called *Kerley B lines* (Figure 34.5). These lines represent fluid in the interlobular septa. If due to heart failure, they indicate pulmonary venous pressures greater than 20 mm Hg. There is a wide variation in clinical correlation at this stage from severe dyspnea without clinical findings to orthopnea or a dry cough. The absence of Kerley B lines does not exclude high pressure. Fluid in the interlobular septa in other portions of the lung fields are called *Kerley A and C lines*. They have a similar but more variable appearance. Kerley B lines are most often associated with acute heart failure. They can occur, however, in other conditions such as mitral stenosis with chronic failure and uncommonly acute viral pneumonias.

Alveolar edema follows the interstitial phase (Figure 34.6). *Alveolar infiltrates* are generally bilateral, perihilar, and symmetric. They can be patchy or cause densely opacified lung fields. Underlying lung diseases can cause variable and asymmetric appearances of pulmonary edema. Edema cannot develop where there is extensive bullous disease as in emphysema or where there is fibrosis. Underlying honeycombing may give a "Swiss cheese" effect with the development of pulmonary edema (Figure 34.7). On the other hand, pulmonary edema can occur on a noncardiogenic basis from causes such as drug intoxications, drowning, toxic inhalations, hypersensitivity pneumonitis, renal failure, and adult respiratory distress syndrome. The cardiac contri-

Figure 34.7
In this case of pulmonary edema, underlying pulmonary parenchymal disease (bullous emphysema) creates asymmetric infiltrates with a "Swiss cheese" or accentuated honeycombed pattern.

Figure 34.6
Extensive alveolar edema is present here with cardiomegaly, a right-sided pleural effusion, a visible horizontal fissure, bilateral perihilar haze, and alveolar infiltrate.

bution to pulmonary edema can be determined by the placement of a Swan-Ganz catheter.

Extensive bilateral symmetric alveolar infiltrates looking just like pulmonary edema can also be seen with severe fulminating pneumonias such as pneumococcal, staphylococcal, or pneumocystis types. Pulmonary hemorrhage and alveolar proteinosis can also mimic congestive heart failure.

Bilateral or right-sided *pleural effusions* can occur at any stage of acute heart failure and may take a long time to resolve. It is not uncommon to have a heart that has returned to its normal size with resolution of the edema and pulmonary venous engorgement but a residual pleural effusion. It is rare to have a purely left-sided effusion with heart failure.

Pulmonary arterial or precapillary hypertension is manifested by *enlargement of the central pulmonary arteries in the hilum* without an associated increase (or even with a decrease) in the size and number of the peripheral vessels (Figure 34.8). In advanced pulmonary arterial hypertension, the hilar vessels may be so large that they mimic adenopathy. In this case, fluoroscopy is useful to demonstrate the pulmonary artery pulsations or "dance." With longstanding hypertension, the right heart fails, resulting in right ventricular enlargement or cor pulmonale. Pleural effusions can occur at this stage as part of a generalized peripheral edema due to right-sided failure.

Pulmonary arterial hypertension can occur as a primary idiopathic disease or as a result of chronic obstructive pulmonary disease, interstitial fibrosis of any cause, and restrictive thoracic wall diseases such as severe kyphoscoliosis. It can occur acutely as a result of severe hypoxemia and acidosis from hypoventilation. The characteristic changes of dilated central pulmonary arteries with abrupt tapering

Figure 34.8
In this case, primary pulmonary hypertension has caused dramatically enlarged hilar vessels with "pruning" of distal vessels, creating a relative radiolucent appearance to the outer lung fields. Because of massive right ventricular enlargement, the left cardiac convexity is formed by a dilated right ventricle.

of second-order peripheral vessels do not give quantitative information regarding the degree of resistance; however, the presence of calcification in the main pulmonary artery indicates long-standing disease.

Pulmonary embolization, if classified by pathophysiology, belongs under the heading of pulmonary arterial hypertension; however, radiographically, it only rarely produces the changes associated with pulmonary artery hypertension. Indeed, in half the cases of pulmonary emboli the chest x-ray will be normal, and in another half, nonspecific changes of decreased lung volume with elevation of the hemidiaphragm and discoid or platelike atelectasis, small pleural effusions, or focal peripheral infiltrates representing areas of hemorrhage and infarct will be seen. The appearance of an infiltrate is sometimes suggestive if it is peripheral, pleural based, and wedge shaped with the apex pointing toward the hilum (*Hampton's hump*). A rare sign is localized oligemia with abrupt vessel cutoff (*Westermark's sign*).

Shunting refers to changes in pulmonary blood flow and volume from congenital cardiac defects. (We are excluding here causes of focal intrapulmonary shunting such as pulmonary emboli and pneumonia.) An estimate of overall blood flow in the lungs can suggest the presence of a left-to-right shunt (with increased flow) or a right-to-left shunt (with decreased flow). Recognition of what is normal or abnormal is a matter of experience rather than specific criteria. Despite the subjectivity, evaluation of *pulmonary blood volume* is an important part of the cardiovascular interpretation of the chest x-ray and should not be overlooked. For example, a patient with an untreated atrial septal defect will have an enlarged right heart, often massively enlarged, and hyperemic lung fields. When the right heart increases in size, the left heart and in particular the left atrium remain normal in size. The size of the heart and degree of hyperemia will depend on the size of the defect. A defect 2 cm or greater will result in significant hemodynamic and radiographic changes.

The most common congenital lesions noted in the adult are atrial septal defect, congenital pulmonary stenosis, and coarctation of the aorta. Atrial septal defect has been discussed above. Pulmonic stenosis in the adult is frequently mild. The radiographic changes include poststenotic dilatation of the pulmonary artery and often predominantly the left main pulmonary artery. The degree of dilatation does not correlate with the degree of obstruction. If long-standing, the artery may calcify. The right ventricle is hypertrophied but generally not dilated and therefore is normal on chest x-ray. Generally, blood flow is also normal, and the lung fields are euvolemic.

Coarctation of the aorta may present in the adult as a previously unrecognized cause of systemic hypertension or with the sequelae of treated disease, which include recurrence of the obstruction, development of aortic valve disease, or progressive atherosclerosis. The coarctation is generally just beyond the origin of the left subclavian artery. With poststenotic dilatation, there is an indentation in the aortic knob. Additional signs include left ventricular enlargement due to hypertension and the development of collateral vessels around the obstruction. This has two effects. First, the dilated vessels may be visible on a chest x-ray; the mammary arteries as serpiginous retrosternal densities on the lateral view or the left subclavian artery overlying the apex of the lung on the PA view. Second, dilated intercostal arteries can erode the undersurface of the ribs, producing "rib notching." These appear as small, bilateral or unilateral, well-defined cortical defects with a sclerotic border on the undersurface of the fourth through tenth ribs.

The Aorta

In the normal young adult, the aortic knob is seen in the PA view and a small portion of the arch on the lateral view. With age, hypertension and atherosclerotic disease, the aorta elongates so that a portion of the descending aorta may be visible on the PA film just above the right atrium and more of the descending aorta is seen on the lateral view. Atherosclerosis and hypercalcemic states in the younger patient cause calcification of the wall. This is usually noted at the knob but can occur anywhere. Aneurysms present as a localized dilation on either the PA or lateral views and may be mistaken for a mediastinal mass. Etiologies include atherosclerotic disease, trauma, and, less commonly today, syphilitic aortitis. Dissecting aneurysms, whether atherosclerotic or traumatic, show widening of the mediastinum with loss of the aortic knob shadow and sometimes pleural fluid. A classic appearance is fluid at the apex of the left lung called the *apical cap*; however, the fluid can accumulate anywhere including the right pleural space. The blood in the mediastinum will push the trachea and esophagus to the right. The latter can be recognized if a nasogastric tube is displaced to the right of its expected course. Another classic but somewhat insensitive sign is a 1 cm or greater separation between intimal calcification and the outer border of the aorta. About 10 to 20% of dissections will have a normal chest x-ray.

Thoracic Cage

Bone diseases such as severe kyphoscoliosis or severe vertebral collapse from multiple myeloma can lead to restrictive lung disease and cardiac compromise. Scoliosis and pectus excavatum can give a false impression of cardiomegaly due to a normal but displaced heart.

References

Braunwald E. Heart disease. Philadelphia: W.B. Saunders, 1988.

Felson B. Chest roentgenology. Philadelphia: W.B. Saunders, 1973.

Fraser RG, Pare JA. Diagnosis of disease of the chest. Philadelphia: W.B. Saunders, 1978.

Kaplan S. The adult with congenital heart disease. Semin Roentgenol 1985;20:151–59.

Milne E. Correlation of physiologic findings with chest roentgenology. Radiol Clin North Am 1973;11:17–47.

Randall P. Thoracic aortic aneurysm. Postgrad Radiol 1983;3:221.

Simon M. The pulmonary veins in mitral stenosis. J Fac Radiol 1958;9:25.